

SUPPLEMENTAL MATERIAL

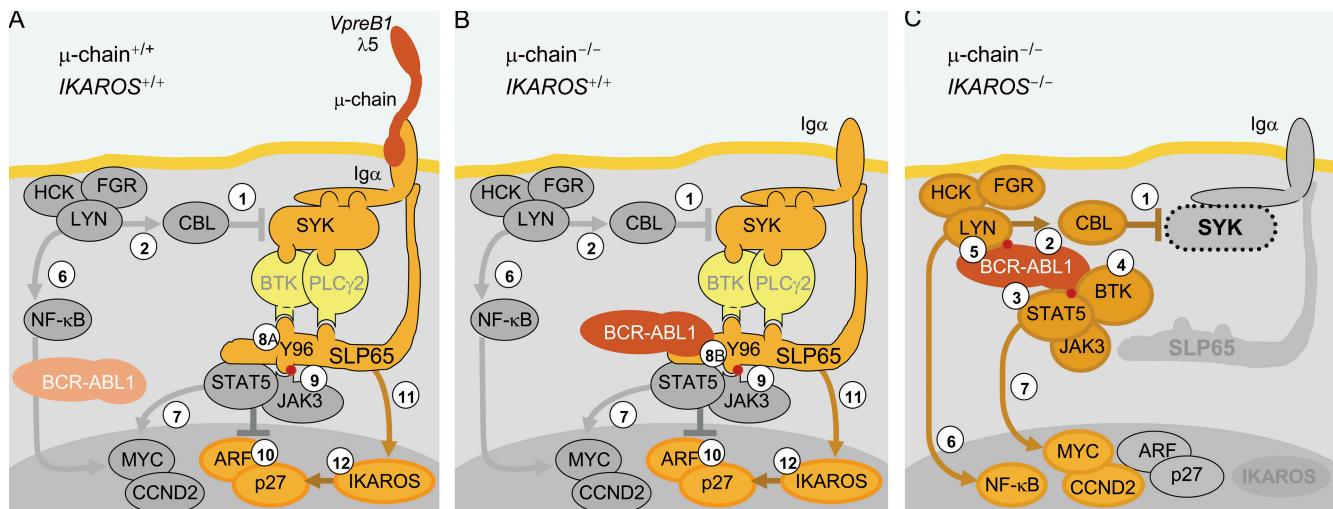


Figure S1. Scenario: the pre-B cell receptor and IKAROS cooperate as tumor suppressors in Ph⁺ ALL. (1) The ubiquitin ligase CBL targets SYK for proteasomal degradation (Ota and Samelson. 1997. *Science*. doi:10.1126/science.276.5311.418). (2) The SRC family kinase LYN activates CBL (Song, H., Zhang, Y.J., Chiang, R.P., Siraganian, and R.J. Hodes. 2007. *J. Immunol.* 178:926–935). (3) BCR-ABL1 activates STAT5-JAK3. (Ilaria, R.L. Jr., and R.A. Van Etten. 1996. *J. Biol. Chem.* doi:10.1074/jbc.271.49.31704). (4) BCR-ABL1 activates BTK, but neither SYK nor SLP65 (Feldhahn, N., F. Klein, J.L. Mooster, P. Hadweh, M. Sprangers, M. Wartenberg, M.M. Bekhite, W.K. Hofmann, S. Herzog, H. Jumaa, et al. 2005. *J. Exp. Med.* doi:10.1084/jem.20042101). (5) Activation of SRC family kinases by BCR-ABL1 is critical for leukemic transformation of pre-B cells (Hu, Y., Y. Liu, S. Pelletier, E. Buchdunger, M. Warmuth, D. Fabbro, M. Hallek, R.A. Van Etten, and S. Li. 2004. *Nat. Genet.* doi:10.1038/ng1343; Ptaszniak, A., Y. Nakata, A. Kalota, S.G. Emerson, and A.M. Gewirtz. 2004. *Nat. Med.* doi:10.1038/nm1127). (6) SRC family kinases drive survival and proliferation of pre-B cells through activation of NF-κB (Saijo, K., C. Schmedt, I.H. Su, H. Karasuyama, C.A. Lowell, M. Reth, T. Adachi, A. Patke, A. Santana, and A. Tarakhovsky. 2003. *Nat. Immunol.* doi:10.1038/ni893; Sprangers, M., N. Feldhahn, S. Herzog, M.L. Hansmann, M. Reppel, J. Hescheler, H. Jumaa, R. Siebert, and M. Müschen. 2006. *Oncogene*. doi:10.1038/sj.onc.1209510). (7) Tyrosine-phosphorylated STAT5 induces up-regulation of MYC and CCND2 and increases pre-B cell proliferation (Tsuruyama, T., T. Nakamura, G. Jin, M. Ozeki, Y. Yamada, and H. Hiai. 2002. *Proc. Natl. Acad. Sci. USA*. doi:10.1073/pnas.112202899). (A, 8) Pre-B cell receptor signaling results in phosphorylation of SLP65 Y96 (Nakayama, J., M. Yamamoto, K. Hayashi, H. Satoh, K. Bundo, M. Kubo, R. Goitsuka, M.A. Farrar, and D. Kitamura. 2009. *Blood*. doi:10.1182/blood-2008-07-166355). (B) Reconstitution of IKAROS expression redirects BCR-ABL1 to phosphorylate SLP65 Y96 (Fig. 7 C). (9) Phosphorylation of SLP65 Y96 is required for SLP65-mediated tumor suppression and the ability of SLP65 to bind and inhibit JAK3 (Nakayama, J., M. Yamamoto, K. Hayashi, H. Satoh, K. Bundo, M. Kubo, R. Goitsuka, M.A. Farrar, and D. Kitamura. 2009. *Blood*. doi:10.1182/blood-2008-07-166355). (10) SLP65 Y96-mediated inhibition of STAT5-JAK3 leads to up-regulation of p27 and induces cell cycle arrest (Nakayama, J., M. Yamamoto, K. Hayashi, H. Satoh, K. Bundo, M. Kubo, R. Goitsuka, M.A. Farrar, and D. Kitamura. 2009. *Blood*. doi:10.1182/blood-2008-07-166355). (11) Pre-B cell receptor/SLP65 signaling induces up-regulation of IKAROS (Ma, S., S. Pathak, L. Trinh, and R. Lu. 2008. *Blood*. doi:10.1182/blood-2007-08-110106; Thompson, E.C., B.S. Cobb, P. Sabbattini, S. Meixlsperger, V. Parelho, D. Liberg, B. Taylor, N. Dillon, K. Georgopoulos, H. Jumaa, et al. 2007. *Immunity*. doi:10.1016/j.jimmuni.2007.02.010). (12) IKAROS induces up-regulation of p27 and cell cycle arrest (Kathrein, K.L., R. Lorenz, A.M. Innes, E. Griffiths, and S. Winandy. 2005. *Mol. Cell. Biol.* doi:10.1128/MCB.25.5.1645-1654.2005).

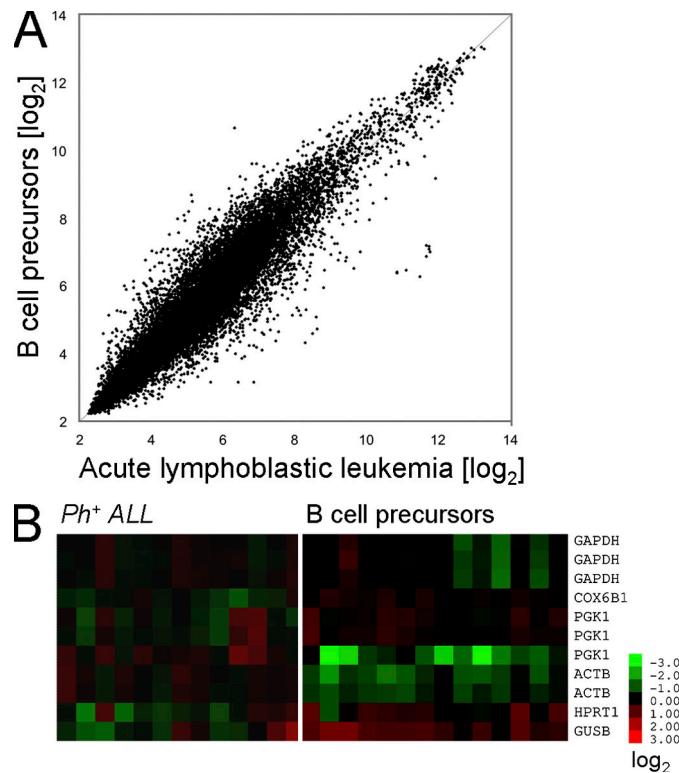


Figure S2. Normalization and validation of metaanalysis of Affymetrix U133A2.0 analysis of gene expression in human Ph⁺ ALL and B cell precursor samples. To investigate expression levels of pre-B cell receptor-related signaling molecules in human Ph⁺ ALL cells, we performed a metaanalysis of publicly accessible Affymetrix U133A2.0 gene expression profiles of 15 cases of Ph⁺ ALL (<http://www.stjuderesearch.org/data/ALL3/rawFiles.html>; Ross, M.E., X. Zhou, G. Song, S.A. Shurtleff, K. Girtman, W.K. Williams, H.C. Liu, R. Mahfouz, S.C. Raimondi, N. Lenny, et al. 2003. *Blood*. doi:10.1182/blood-2003-01-0338) and normal human B cell precursors, including pro- and pre-B cells, as well as immature B cells (<http://130.161.42.18/>; Fig. 1 C; van Zelm, M.C., M. van der Burg, D. de Ridder, B.H. Barendregt, E.F. de Haas, M.J. Reinders, A.C. Lankester, T. Révész, F.J. Staal, and J.J. van Dongen. 2005. *J. Immunol.* 175:5912–5922.). (A) A scatterplot of the two datasets after processing with the RMA algorithm is shown. All B cell precursor subsets (12 samples) were grouped in the “B cell precursor” class, and three randomly picked samples of each ALL subtype were grouped to *BCR-ABL1* ALL cases (12 cases in total). Dots represent probesets of the U133A2.0 GeneChips used as values calculated by \log (base 2) ratios averaged over phenotype classes. Genes that are equally expressed in both groups are represented by dots lying on the diagonal. (B) Heat map of selected housekeeping genes is shown. Similar expression values for housekeeping genes between the ALL and B cell precursor groups is taken as an indication of correct normalization. Probesets with “absent” calls are excluded from the analysis.

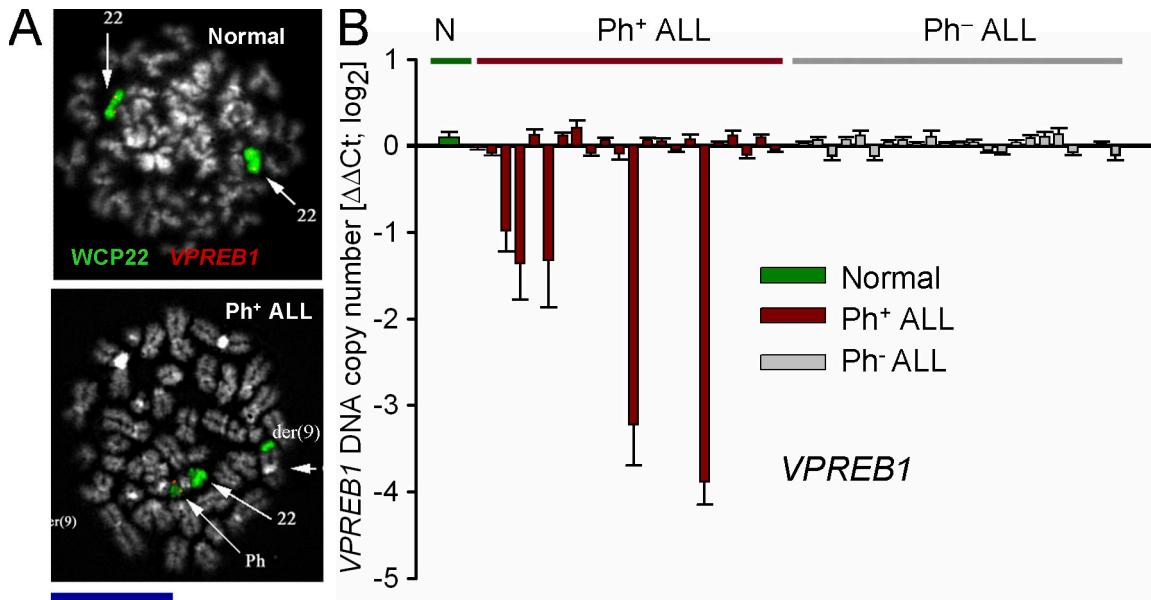


Figure S3. Verification of VPRE-B1 gene deletions in Ph⁺ ALL. Deletions of the *VPREB1* locus were verified by FISH (A) and by genomic quantitative PCR (B). These deletions are located in a cluster on chromosome 22q11 between basepairs 20913571 and 20931814. DNA was isolated from 22 cases of Ph⁺ ALL and 22 cases of Ph⁻ ALL. Quantitative PCR of *VPRE-B1* DNA was normalized using *MYCN* as a control. *VPRE-B1* copy number changes are shown in Ph⁺ ALL and Ph⁻ ALL as mean of triplicate measurements (+ SEM). Bar, 10 μ m.

Fluorescence in situ hybridization (FISH). Chromosome preparations from bone marrow cells were hybridized in situ with 1 μ g of probe labeled by nick translation. Hybridization was performed at 37°C in 2X SSC, 50% (vol/vol) formamide, 10% (wt/vol) dextran sulfate, 5 μ g COT1 DNA (Bethesda Research Laboratories), and 3 μ g sonicated salmon sperm DNA in a volume of 10 μ l. Posthybridization washing was performed at 60°C in 0.1X SSC (three times). In cohybridization experiments, the probes were directly labeled with Fluorescein and Cy3. Chromosomes were identified by DAPI staining. Digital images were obtained using a DMRXA epifluorescence microscope (Leica) equipped with a cooled xcharge-coupled device camera (Princeton Instruments). Cy3 (red; New England Nuclear), fluorescein (green; Fermentas Life Sciences), and DAPI (blue) fluorescence signals, which were detected using specific filters, were recorded separately as grayscale images. Pseudocoloring and merging of images were performed with Adobe Photoshop software. The whole chromosome paints (WCP) used for chromosomes 22, derived from flow-sorted chromosomes, was a gift from the Wellcome Trust Sanger Institute, Cambridge, England (N. Carter). A probe specific for the *VPREB1* gene (11,615 bp) was obtained as a product of LONG-PCR, performed using TaKaRa LA Taq DNA polymerase (Cambrex Bio Science), by designing an appropriate primer pair (Table S1) according to the latest release (March 2006) of the University Santa Cruz Genome Browser (<http://genome.ucsc.edu/>).

Real-time quantitative genomic PCR. For validation of FISH and SNP mapping analyses, *VPREB1* gene copy numbers relative to *MYCN* were determined by quantitative genomic PCR in 22 Ph⁺ ALL cases and 22 Ph⁻ ALL cases. These measurements were performed on bone marrow samples from Ph⁺ ALL and Ph⁻ ALL with >80% blast infiltration. Germline DNA from normal bone marrow (7 donors) was used as a control. Quantitative genomic PCR was performed using SYBR Green PCR master Mix (Applied Biosystems) and 100 ng of extracted genomic DNA for each reaction. Taqman SYBR Green assays were performed using a 7900 Real-Time PCR system and 7900 System Software (Applied Biosystems). The quantitative PCR thermal protocol consisted of 50°C for 2 min, followed by 95°C for 10 min, and then 40 cycles of 95°C for 1 min and 60°C for 1 min. *MYCN* was used as control gene.

Table S1A. IGHM gene rearrangements in human B cell precursors

Pro-B I.8	ARDRHI*#FDYW
Pro-B I.9	CAKDLRRGHFDWSLEYDSW
Pro-B II.1	CARSGYSYARRGYYYYGMDVW
Pro-B II.3	CTTVHPSCYSGYGGYGCYGYGYTTSM*W CARDLVTTSPYYMDVW
Pro-B II.10	CARSGYSYARRGYYYYGMDVW
Pro-B II.15	CAKDAGSGYFDSW
Pro-B II.17	CARWRNLEWYPW
Pro-B III.3	TRTENYYDSTAYS
Pro-B III.9	CFERGYYGLLGPWHLVIIRSTSD#HDYYGMDVW CARSRGYYY#*YGMGVW
Pro-B III.13	CAREKYSGYDLDYYYYGMDVW
Pro-B III.15	CAKVVMMVVMVMMVVMVVMVVMVMT#DAW
Pro-B IV.3	CATPTRQ*WSKPLFDYW
Pro-B IV.7	CAKEGQGVLPYDTGDYW
Pro-B IV.9	CARSGYSYARRGYYYYGMDVW
Pro-B IV.11	CVERERFSSGGY*LLQ
Pro-B IV.15	CTRDAVVDTIF*RMAS#DYW
Pro-B IV.20	CARDSLPSLPMPEGGSFW
10/17 pro-B cells 58.8%	10/19 V _H DJ _H gene rearrangements potentially functional 52.6%
Pre-B I.2	CARRPAAGTGWFDPW
Pre-B I.11	CARSNWGTDPDFDW
Pre-B I.14	SARIIVYDSRDLYRLGWYFDLWGRG
Pre-B II.12	CARERITMIVIMDVW
Pre-B II.16	ARGTV*KTTSSE CAREGGTIFGVLYFDYW
Pre-B II.20	CARGLEGNSGSFHFDYW
Pre-B III.2	CERF*VWIFGVVIIYAP*YYCGMDVW CARAHVGASGALDYW
Pre-B III.6	CAGGAVFDYW
Pre-B III.14	CALRLGVAGTGDDAFDIW
Pre-B III.17	CAKDLYYGGYGGYGCYGYGFYVDAW
Pre-B III.20	CARPALPGGQAYGMDVW
Pre-B IV.3	CARVTTVR *#YYYYYYYYMDVW
Pre-B IV.4	CAKDPTPRSSLFDYW
Pre-B IV.11	CVRADLSLTAGGHFDSW
Pre-B IV.14	CAKVADQQL#YFDSW
Pre-B IV.20	CAKDSSCYSGCLYGMGVW
14/16 pre-B cells 87.5%	14/18 V _H DJ _H gene rearrangements potentially functional 77.8%
IB I.15	CARGVVVAATTWDYW
IB I.17	CARRVEMATKGAFDIW
IB I.18	CARDQPNFGVVRGHYYYYGMDVW
IB II.3	CASRYCIYQLLWGPCL#DVW
IB II.5	CAREPNNSGSYGFDPW
IB II.12	CAVGRTYLDYW
IB II.17	CARDRRFDWFGLDYYYYMDVW
IB II.20	CARDLQVATTVTTSPYYYYMDVW
IB III.2	CARTTMIVVVIAGDSTP
IB III.3	CARSGYSYARRGYYYYGMDVW
IB III.4	CARDPLYGDYDPLEYGMDVW
IB III.5	CARQPDTAGFWFDPW
IB III.6	CASPNNYGGSGNPYPYYYYGMDVW
IB III.7	CARDRGYGDYGLYFQHWGQGTLSPLSPQVSLLSGDSEPGV
IB III.15	CARGTNAIVPAAISSSWYDVRFDYW
IB IV.5	CARHLL*HGW*WTS*SIYYYYYYV#DVW
IB IV.8	CARALGYVVVVPAAMRGWGHYGMGVW
15/17 immature B cells 88.2%	15/17 V _H DJ _H gene rearrangements potentially functional 88.2%
39/50 Normal B cell precursors 78.0%	39/54 V _H DJ _H gene rearrangements potentially functional 72.2%

Notes: Gray shades denote non-functional V_HDJ_H gene rearrangements or cases lacking coding capacity for a pre-B cell receptor
 *Stop codon
 # frame shift

Table S1B. IGHM gene rearrangements in Ph⁺ ALL

Ph ⁺ ALL I	I PADCGGDC*GGWFDPWGQG	Ph ⁺ ALL XLV	CARPHY#DAFDIW
Ph ⁺ ALL II	CAGDGYN#YYYYGMDVW	Ph ⁺ ALL XLVI	CSRGRSIAAKIPLL##YFDYW
Ph ⁺ ALL III	CARGGTGGDCYH#SSYWYFDLW	Ph ⁺ ALL XLVII	GTTQAGSTP#
Ph ⁺ ALL IV	WLQLF#LTTGARE	Ph ⁺ ALL XLVIII	CARV#GDYW
Ph ⁺ ALL V	CARAPDPPSMAVRGKGLRV*FPYYYGM	Ph ⁺ ALL XLIX	CAKDXPRILWW*LLFR
Ph ⁺ ALL VI	EG*WEPPMLLISGAKG	Ph ⁺ ALL L	CARHP*GIAAFCI##
Ph ⁺ ALL VII	CARDSLRYFDWLIG#DYW	Ph ⁺ ALL LI	LLYP#YGMDVW
Ph ⁺ ALL VIII	CARGSGY##FDYW	Ph ⁺ ALL LII	CTTRRALVVPAA#YYG
Ph ⁺ ALL IX	CAKDQAWE##YFD#W	Ph ⁺ ALL LIII	CARDRIS*YCXGGSCRERNYY
Ph ⁺ ALL X	CARPL#CFDYW	Ph ⁺ ALL LIV	CARDGPGYCSGGSC*WVGITM
Ph ⁺ ALL XI	CAPL*LERR#DYW	Ph ⁺ ALL LV	HLLPERNS*TYMVRG
Ph ⁺ ALL XII	CA*GVLRYFDWLLYAPA#NWFDPW	Ph ⁺ ALL LVI	AVGVH*DIVVVPAAIHYYGYM
Ph ⁺ ALL XIII	CARDGPGRRLRLGPGNPGRHLRL*GRIR	Ph ⁺ ALL LVII	CAR*PFDPW
Ph ⁺ ALL XIV	CARDLGGSGS**P*VNWFDPW		CARVDRGG*LLRYGRLL
Ph ⁺ ALL XV	CARI##FDPW		
Ph ⁺ ALL XVI	VH*DIVVVPA		
Ph ⁺ ALL XVII	CKPSLGDFWSR1DYW		
Ph ⁺ ALL XVIII	CVRDGDTSWSFDYW		
Ph ⁺ ALL XIX	CARDPSGWCGGDS*HH#FDYW		
Ph ⁺ ALL XX	CASGEKPRSTTVV#FYYYYYGM		
Ph ⁺ ALL XXI	CART#SSTS YDGM		
Ph ⁺ ALL XXII	CAEVPLIF*LVI IFDYW		
Ph ⁺ ALL XXIII	CARAVSH#YNWFEGPG		
Ph ⁺ ALL XXIV	CATLA#SGWYRYFDLW		
Ph ⁺ ALL XXV	CARDMGSERWIQLWLL*CPF#FDYW		
Ph ⁺ ALL XXVI	VYATSPYYYYGM		
Ph ⁺ ALL XXVII	CARGG*GAAAGTR#NWFDPW		
Ph ⁺ ALL XXVIII	CARDTADVL**YP#FDYW		
Ph ⁺ ALL XXIX	CARALGITMIAVALGE#F		
Ph ⁺ ALL XXX	CARPGRFSSG WYSAFDI#		
Ph ⁺ ALL XXXI	ASSGYSYWGQGTTVTVSSV*TCRK		
Ph ⁺ ALL XXXII	CARHTVRETSPEPV*NPPHRR		
Ph ⁺ ALL XXXIII	CRMSC*RLTPQGPA		
Ph ⁺ ALL XXXIV	CARHTVRETSPEPV*NPCRMSC*RLTPQGPA		
Ph ⁺ ALL XXXV	CARDPPSITMIVVVIMG#DAFDIW		
Ph ⁺ ALL XXXVI	CARHQQTVRGPLDPW		
Ph ⁺ ALL XXXVII	CAHSPRDPRG**WLYGSNSRYYFDYW		
Ph ⁺ ALL XXXVIII	CAHSPRDPRG**WLLLGI		
Ph ⁺ ALL XXXIX	CTRECITVRETAGYSSWFDF*		
Ph ⁺ ALL XL	CASQIL*WW*LPI#GAFDIW		
Ph ⁺ ALL XLI	CAYQKNLQIYTF*KVVI*RPSLTCTKCRSMRSSQPRSPSPQ		
Ph ⁺ ALL XLII	CARAGSGDYGLYYYYYGM		
Ph ⁺ ALL XLIII	DVWGQGTTVTVSS		
Ph ⁺ ALL XLIV	CARASGNPTYYYYMDVWGKGTTVTVSS		
Ph ⁺ ALL XLV	CVKPMGPYREAFDIWGQG		
Ph ⁺ ALL XLVI	LVRELPGGVCY#TGTTTTVWTSGAKG		
Ph ⁺ ALL XLVII	CTRVARCWCMLYRYYYYGM		
Ph ⁺ ALL XLVIII	DVWGQGTLVTVSS		
Ph ⁺ ALL XLIX	CARRDCSSTSCYTSDDYYGM		
Ph ⁺ ALL XLX	DVWGQGTLVTVSS		
Ph ⁺ ALL XLXI	CTRVARCWCMLYRYYYYGM		
Ph ⁺ ALL XLII	#CEMG#EQPVYYGM		
Ph ⁺ ALL XLIII	DVWGQGTLVTVSS		
Ph ⁺ ALL XLIV	CARWAGTTG#YYGM		
Ph ⁺ ALL XLV	CARVRYYDSSGYYHYYGM		
Ph ⁺ ALL XLVI	DVWGQGTLVTVSS		
Ph ⁺ ALL XLVII	CARER*KGLRGVL*LLGPG		
Ph ⁺ ALL XLVIII	CARELSRRYSDLWGRGTTVTVS##		
Ph ⁺ ALL XLIX	CARGVLLLGPGNPGHRL		
Ph ⁺ ALL XLX	CAHRSGVLLLGPGNPGHRL#		
Ph ⁺ ALL XLXI	ITVLWW*LLLRAVWTSGAKG		
Ph ⁺ ALL XLXII	*CEG*KGLRGVL*LLGPG		
Ph ⁺ ALL XLXIII	CARDKRDYGGYFDYW		
Ph ⁺ ALL XLXIV	TV#EMGGV#LTTGARE		
Ph ⁺ ALL XLXV	CARVWYQLMPRDW		
Ph ⁺ ALL XLXVI	CVRDGDTSWSFYW		
Ph ⁺ ALL XLXVII	CPEYSSGYGLPGPYGMVW		
Ph ⁺ ALL XLXVIII	CARSRAVTT*#YFDYW		
Ph ⁺ ALL XLXIX	CAGSTFLDWLLL##AFDW		
Ph ⁺ ALL XLX	CAGG#NWFDPW		
Ph ⁺ ALL XLXII	CVRESSYDFWSGY##FDPW		
Ph ⁺ ALL XLXIII	CARDRWV*QQLVRPLRLWGQGTLVT		

Potentially productive V_HDJ_H gene rearrangements:

10/57 Ph⁺ ALL cases 17.5%
14/82 V_HDJ_H gene rearrangements 17.1%

Notes: Gray shades denote non-functional V_HDJ_H gene rearrangements or cases lacking coding capacity for a pre-B cell receptor
 *Stop codon
 # frame shift

Table S1 C. Statistical model for probability of functional and non-functional *IGHM* V_HDJ_H gene rearrangements

Assuming random distribution of in-frame (1 in 3; [0.33]) and out-of-frame (2 in 3; [0.67]) V_HDJ_H -gene rearrangements and random acquisition of stop codons (3 in 64; [(1-61/64)⁴ or 0.17 for an average V_HDJ_H junction of 4 codons]), we expect the following frequencies of nonfunctional *IGHM* gene rearrangements:

One *IGHM* allele rearranged:

If only one *IGHM* locus is rearranged in a pre-B cell, the probability of a nonfunctional junction based on random distribution of out-of-frame rearrangements (0.67) and acquisition of stop codons (0.17) is ~72% [1–0.33 × (1–0.17)].

Both *IGHM* alleles rearranged:

If both *IGHM* alleles are rearranged, we expect a frequency of ~52% [0.72²] of cells/leukemia clones lacking coding capacity for a pre-B cell receptor. Based on these assumptions, we calculated the expected frequency of cells/ Ph^+ ALL clones lacking coding capacity in the absence of positive or negative selection for expression of a functional pre-B cell receptor (gray lines, Fig. 1 A).

Table S2: Characterization of progressive leukemic transformation in *BCR ABL1*-transgenic mice

	Wild type	Pre-leukemic	Leukemic	Post-AMN107
White blood counts [$\times 10^9/l$; n=5]	6.0 ± 1.8	15.8 ± 5.2	146.4 ± 56.9	11.4 ± 5.2
Peripheral blood BCPs [%CD19 $^+$ AA4.1 $^+$; n=5]	0.2 ± 0.1	4.2 ± 7.4	78.4 ± 7.0	1.4 ± 0.7
Splenic weight [mg; n=4]	82.9 ± 15.5	106.3 ± 27.4	289.7 ± 80.8	125.7 ± 19.5

BCP, B cell precursor

Table S3. Leukemic growth characteristics of *SLP65*^{-/-} BCR-ABL1 ALL cells in the presence or absence of Slp65 reconstitution in vivo

	Irradiation control	GFP	<i>S/p65-IRES-GFP</i>
White blood counts [x 10 ⁹ /liter; n = 3]	5.1 ± 1.9	122.4 ± 21.5	9.8 ± 4.4
Leukemic blasts [%CD19 ⁺ GFP ⁺ ; n = 3]	0	85.1 ± 11.8	0.4 ± 0.3
Splenic weight [mg; n = 3]	78.6 ± 16.4	152.8 ± 55.0	118.4 ± 21.1

Table S4. Sequences of oligonucleotide primers used

Single-cell PCR for <i>IGHM</i> gene rearrangements (human)	
V _H 1	5'-CAGTCTGGGCTGAGGTGAAGA-3'
V _H 2	5'-GTCCTRCGCTGGTGAAACCCACACA-3'
V _H 3	5'-GGGGTCCCTGAGACTCTCCTGTGCAG-3'
V _H 4	5'-GACCCTGTCCCTCACCTGCRCGTGTC-3'
V _H 5	5'-AAAAAGCCCAGGGAGTCTCTGARGA-3'
V _H 6	5'-ACCTGTGCCATCTCGGGGACAGTG-3'
3'J _H 1.2.4.5	5'-ACCTGAGGAGACGGTGACCAGGGT-3'
3'J _H 3	5'-ACCTGAAGAGACGGTGACCATTGT-3'
3'J _H 6	5'-ACCTGAGGAGACGGTGACCCTGGGT-3'
5'J _H 1.4.5	5'-GACGGTGACCAGGGTKCCCTGGCC-3'
5'J _H 2	5'-GACAGTGACCAGGGTGCCACGGCC-3'
5'J _H 3	5'-GACGGTGACCATTGTCCCTGGCC-3'
5'J _H 6	5'-GACGGTGACCGTGGTCCCTKGCC-3'
Quantitative genomic PCR (human)	
VPREB1_F	5'-ATTTCTCACAAATCAGACAAGAGCCA-3'
VPREB1_R	5'-CTCAGAGATGCTCAAATACCCCCT-3'
MYCN_F	5'-GGAAAGAACCCCTCAGTCG-3'
MYCN_R	5'-AAGTCATCTCGTCCGGTA-3'
Long distance PCR primers to generate FISH probe	
VPREB1_F	5'-TGTTATTGCACAAACATCCACAA-3'
VPREB1_R	5'-AAAGGCTGTGTCATGGGAAG-3'
Clonality and spectratyping analysis (mouse)	
V _H J558_F	5'-AAGGCCACACTGACTGTAGAC-3'
C _μ _R	5'-TGGCCACCAGATTCTTATCAG-3'
C _μ -FAM_R	5'-AGACGAGGGGAAGACATTG-3'
Quantitative RT-PCR (mouse; <i>BCR-ABL1</i> human)	
Slp65_F	5'-GAAGGGACTACGCATTAGACAG-3'
Slp65_R	5'-GCATCACATACATCTCGGAGT-3'
BCR-ABL1_F	5'-ATCGTGGCGTCCGCAAGAC-3'
BCR-ABL1_R	5'-GCTCAAAGTCAGATGCTACTG-3'
Hprt_F	5'-GGGGGCTATAAGTTCTTG-3'
Hprt_R	5'-TCCAACACTTCGAGAGGTCC-3'
Ighm_F	5'-CATCTAAAACCAATGAGG-3'
Ighm_R	5'-GGGCACTGGTCACATACTTC-3'
Ikaros_F	5'-ATGTACCCAGTCATTAAGGAAGA-3'
Ikaros_R	5'-TCTCATAGTTGGCACTGTCTAG-3'